# 1

# Bridging the Gap: A Consilient Methodology

A balanced perspective cannot be acquired by studying disciplines in pieces; the consilience among them must be pursued. Such unification will be difficult to achieve. But I think it is inevitable. Intellectually it rings true, and it gratifies impulses that arise from the admirable side of human nature. To the extent that the gaps between the great branches of learning can be narrowed, diversity and depth of knowledge will increase. They will do so because of, not despite, the underlying cohesion achieved. The enterprise is important for yet another reason: It gives purpose to intellect. It promises that order, not chaos, lies beyond the horizon. Inevitably, I think, we will accept the adventure, go there, and find what we need to know.

—Edward O. Wilson, "Back from chaos," *Atlantic Monthly,* March 1998

Over the centuries, science has been the cornerstone of the majority of advances in human well-being, including Hippocrates's initial inquiries into the nature of contagion and human health, Charles Darwin's hypotheses on the evolution of species, Louis Pasteur's development of antimicrobial vaccines, and Robert Fogel's thesis that reductions in morbidity and mortality impelled the industrialization of the United Kingdom.[1]

Recent research has focused on the study of complex relationships between political systems and influences on state capacity such as environmental change, resource scarcity, population, and migration.[2] In this chapter, I discuss methodological principles for the study of complex systems which include health (and specifically infectious disease, a heretofore unexamined major determinant of state capacity).

In this book I test the hypothesis that increasing levels of infectious disease exert a negative effect on state capacity, such that increases in disease prevalence result in correspondingly diminishing values of state capacity.

Thus, I seek to both understand the causal role that disease plays in determining state capacity, and determine the causal relations between the two variables. I also seek the answers to the following two questions: Can infectious disease negatively affect state capacity by generating political, economic and social instability? If so, how does infectious disease contribute to political instability and underdevelopment?

The biologist Edward O. Wilson concedes that the narrow compartmentalization of science in the nineteenth and twentieth centuries provided many benefits to society, but he bemoans the modern lack of consilience as detrimental to the greater pursuit of scientific knowledge in the years to come. Consilience is defined as the "jumping together of knowledge as a result of the linking of facts and fact-based theory across disciplines to create a common groundwork of explanation."[3] Francis Bacon, who took all knowledge to be his province, also recognized the need for the practitioners of divergent scientific disciplines to communicate their findings across the artificial boundaries between the branches of human knowledge.[4] As the Enlightenment thinkers of seventeenth- and eighteenth-century Europe understood, there is a profound need to seek scientific insight in the form of consilience at the nexus points where the disciplines meet. If we reject consilience, we risk continuing the fragmentation of knowledge; indeed, we risk creating a scientific "Tower of Babel" wherein we are incapable of communicating across disciplines.

As figure 1.1 illustrates, pathogenic microbes exist independently throughout the earth's biosphere, with the vast majority of them present in the zoonotic pool and outside of the human ecology. In a very real way these pathogens are independent variables and are exogenous to the state; they are truly global phenomena, existing at the system level. These pathogens may cross over from the zoonotic reservoir into the human ecology at any time with emergence being governed largely by the principles of chance zoonotic transmission and microbial evolution.

After pathogenic agents enter the human ecology (and become endogenized within human societies), their effects are augmented by intervening variables that I call *disease amplifiers* (DAs). These DAs generate changes in viral traffic that result in emerging and re-emerging infectious diseases (ERIDs). Thus, ERIDs are a product of the synergy between the inde-

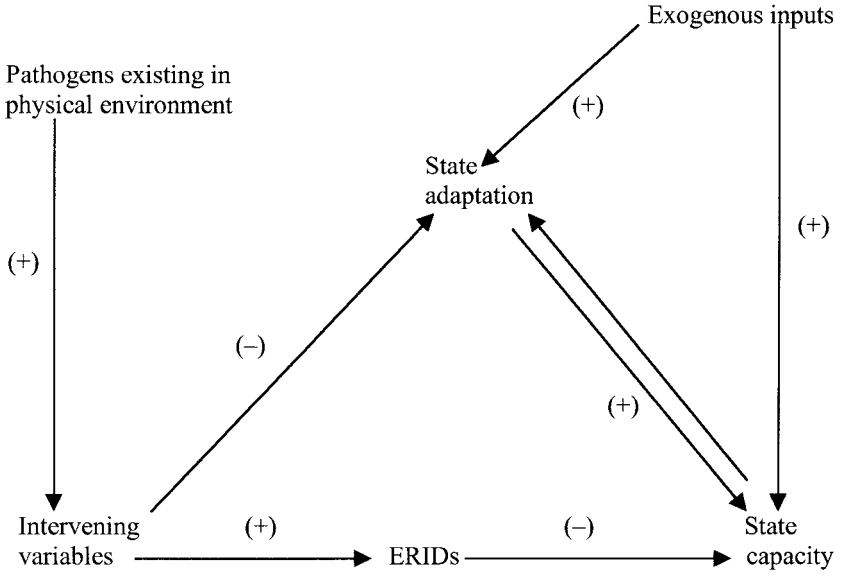


**Figure 1.1**
Probabilistic relations among variables.

pendent variable (pathogens) and intervening variables (such as global environmental change). These ERIDs may, in turn, have pervasive negative effects on state capacity (the dependent variable). Those effects may range from poverty to social and political instability.

States and societies may use adaptive resources to mitigate the effects of disease on state capacity. A state's ability to adapt is limited by several factors. First, the initial level of state capacity will determine the scale of the adaptive resources that may be mobilized to deal with the ERID problem. States with higher initial capacity will therefore have greater technical, financial, and social resources to adapt to crises. Furthermore, state adaptation will be affected by exogenous inputs of capital and by the social and technical ingenuity of international and non-governmental organizations. Finally, state adaptation may be compromised by certain outcomes generated by intervening variables, such as war, famine, and ecological destruction.

Exogenous inputs take the form of inputs of capital, technology, and ingenuity into the state from external sources such as international organizations and bilateral foreign aid. Exogenous inputs such as capital

infusions (from the World Bank and other sources) also directly affect the resources available to a state when responding to crises, and therefore augment the efficacy of adaptation responses.

There is a positive association between state capacity and state adaptation because greater initial capacity means that there are more human, economic, and technical resources endogenized within the state that can then be mobilized to deal with various crises. The lower the initial value of state capacity (SC), the lower the amount of resources that can be mobilized to offset the crisis. This relationship operates in a reciprocal spiral such that greater initial capacity leads to greater adaptive ability, which should in turn reduce the ERID-induced loss to state capacity. Thus, in general, states that have lower SC when diseases afflict them suffer much greater SC losses than states with high initial SC. The only means by which states with lower SC can ameliorate the effects of disease is through exogenous inputs that give low-SC states both greater resources to mobilize and advanced tactical knowledge to deal with the crisis.

Although other relations between variables in the model are also important, in-depth examination of these associations is beyond the scope of this book. The relationship between pathogens in the state of nature and intervening variables is not examined for several reasons:

- Pathogens in the state of nature are generally assumed to be static in population size.
- The potential lethality and transmissibility of these pathogens is unknown until they cross over as zoonoses, are affected by intervening variables, and become ERIDs.
- The manner in which exogenous pathogens become endogenized within the human ecology has been extensively documented by epidemiologists.[5]

Research on these causal relationships is best left to microbiologists, epidemiologists, and public health scientists. I will, however, address the effects of global environmental change on projected and current shifts in disease prevalence, infectivity, and lethality (and concurrent shifts in vector distribution and behavior) in chapter 4.

In the work reported here, I did not test other certain relationships outlined in the causal diagram. Specifically, I did not analyze the relationship

between the intervening variables and the dependent variable, outside of the context of infectious disease. Variables such as war and natural disasters undoubtedly have an independent and logically negative effect on state capacity, but these questions fall outside of the purview of this study. Similarly, the effects of war, environmental degradation, disasters, and migration will undoubtedly have negative effects on state adaptation, but these questions too are beyond the scope of the present book.[6] Such questions are, however, excellent avenues for further research. Finally, I did not test the relationship between exogenous inputs and state capacity. This relationship is very much in need of elaboration, although researchers working on the State Capacity Project of Peace and Conflict Studies at the University of Toronto have begun to untangle the problem.

**The Dependent Variable**

The political scientist Thomas Homer-Dixon has developed a coherent and comprehensive definition of state capacity, which I adopt with minor revisions.[7] He defines the state as "the government, including the center, provincial, and local levels." The term *capacity* generally refers to power and/or capability. Thus, *state capacity* refers to the capability of government. I define state capacity as one country's ability to maximize its prosperity and stability, to exert *de facto* and *de jure* control over its territory, to protect its population from predation, and to adapt to diverse crises. This definition of state capacity corresponds roughly to Homer-Dixon's multi-dimensional definition of state capacity, which consists of one set of variables that measures the state's intrinsic characteristics and a second set that measure relations between state and society. These variables are laid out in table 1.1.

State capacity includes the concept that states are entities that evolve over time.[8] This evolution occurs because of the changing factors that affect state power: land, resources, population, health, technology, human capital, prosperity, and so on. Thus, when we quantify state capacity, we should attempt to measure changes in value on a continuous scale.

For the purposes of this book, I have ranked these attributes of state capacity as follows, in order of decreasing importance:

**Table 1.1**
Indicators of state capacity identified by Project on Environmental Scarcities, State Capacity and Civil Violence, University of Toronto (http://utl1.library.utoronto.ca/www/pcs/state/keyfind.htm).

| | |
|---|---|
| **Indicators of the state's (or its components') intrinsic characteristics** | |
| Human capital | The technical and managerial skill level of individuals within the state and its component parts. |
| Instrumental rationality | The ability of the state's components to gather and evaluate information relevant to their interests and to make reasoned decisions maximizing their utility.[a] |
| Coherence | The degree to which the state's components agree and act on shared ideological bases, objectives, and methods; also, the ability of these components to communicate and constructively debate ideas, information, and policies among themselves. |
| Resilience | The state's capacity to absorb sudden shocks, to adapt to longer-term changes in socio-economic conditions, and to sustainably resolve societal disputes without catastrophic breakdown. The opposite of brittleness. |
| **Indicators of relations between the state (or its components) and society** | |
| Autonomy | The extent to which the state can act independently of external forces, both domestic and international, and coopt those that would alter or constrain its actions. |
| Fiscal resources | The financial capacity of the state or of a component of the state. This capacity is a function of both current and reasonably feasible revenue streams as well as demands on that revenue. |
| Reach and responsiveness | The degree to which the state is successful in extending its ideology, socio-political structures, and administrative apparatus throughout society (both geographically and into the socio-economic structures of civil society); the responsiveness of these structures and apparatus to the local needs of the society. |
| Legitimacy | The strength of the state's moral authority—the extent to which the populace obeys its commands out of a sense of allegiance and duty, rather than as a result of coercion or economic initiative. |

a. *Utility* may be defined locally; i.e., it may reflect the narrow interests of the component and not the broader interests of the state or society.

fiscal resources
human capital
reach and responsiveness
resilience
legitimacy
autonomy
coherence
instrumental rationality.[9]

This ranking emphasizes the primacy of fungible economic power and the importance of human capital and adaptive ability in dealing with the problematic trans-boundary and internal issues of the post-Cold War era, such as global environmental degradation, crime, weapons proliferation, ethnic violence, and pathogen proliferation. Although the factors listed above are all captured by the definition of state capacity, I will operationalize state capacity by using a limited set of empirical indicators.

State capacity is the capability of government, and its level determines the state's ability to satisfy its most important needs: survival, protection of its citizens from physical harm as a result of internal and external predation, economic prosperity and stability, effective governance, territorial integrity, power projection, and ideological projection. Aside from this normative definition, which articulates the dynamic nature and needs of the state, the notion of state capacity should be quantifiable such that we can determine diachronic variance in the value of SC, determine the relative SC of states with respect to one another, and measure correlations between empirical indicators of SC and other parameters (population, resource scarcity, health, technology, environmental degradation) that may affect the value of SC.

Then how can we quantify state capacity in a meaningful way? One answer is to develop a core set of cross-national statistical indicators of SC that may then be correlated against diverse independent variables. To that end, I will correlate the following five statistical indicators for SC against two proxies for ERIDs in order to determine the empirical associations between SC and infectious disease. All indicators of SC are logically valid measures of the performance of government functions.

**Gross national product, per capita (current prices, 1980 US dollars)** measures the total value of goods and services produced by the state on an annual basis. The sum is divided into a per capita measure and standardized for current prices. This is a logically valid measure of SC because high values of this variable require an effective regulatory apparatus. This variable measures such aspects of state capacity as fiscal resources, autonomy, reach and responsiveness, resilience, human capital, and legitimacy.

**Government expenditure (standardized currency, per capita)** measures the total fiscal outlay of the state on the provision of services (e.g. education, health care) to its population on an annual basis. This is a logically valid measure of SC because the more a state spends, the more it is able to generate a revenue stream and it is able to fund a greater number of programs. This variable measures such aspects of state capacity as reach and responsiveness, legitimacy, resilience, and human capital.

**School enrollment ratio, secondary** measures the percentage of the total population of possible secondary school attendees actually receiving secondary education on an annual basis. This is a logically valid measure of SC because education is a core state function and it is expensive. This variable measures such aspects of state capacity as human capital, legitimacy, resilience, reach and responsiveness, fiscal strength, and autonomy.

**Net long-term capital inflow (standardized currency, per capita)** measures the influx of economic capital into the state from exogenous sources over time. It is reasonable to assume that rational investors will seek to put their capital into politically stable and economically productive societies, and thus this variable indicates a measure of state stability and prosperity. This indicator also gives an idea as to external perceptions of state stability. This indicator is a logically valid measure of SC, because countries with low SC cannot guarantee a stable investment climate and a decent rate of return. This indicator measures such aspects of state capacity as fiscal resources, resilience, reach and responsiveness, autonomy, and legitimacy.

**Military spending per soldier, per capita (standardized currency)** measures the government's annual fiscal outlay for defense. The aggregate

amount is then divided by the number of soldiers in the defense forces, and then the value is adjusted so that it reflects a per capita ratio. The per capita, per soldier ratio allows a relative ranking of the amount spent on the training of soldiers and expenditures on weapons systems. High spending per soldier per capita is an indication of high-tech, capital-intensive, and training-intensive armed forces that can only be created and maintained by states that possess high state capacity. This is a logically valid measure of SC because only a state with high SC can afford to fund an efficient, high-quality defense.

These SC indicators provide us with a large data set for the period from the early 1950s to 1991. This allows us to analyze the diachronic associations between variables in order to generate conclusions about the evolutionary path of individual states. It also provides us with enough data so that we can run diachronic correlations to examine the significance of the association between variables. These five SC indicators, the data from 1950 to 1991, and the 20 countries in the sample provide us with a rich set of data points that increase both the significance and certainty of our correlations and the inferences we draw from them. Ultimately, we will be able to run multivariate regressions controlling for the independent variable (e.g., infant mortality), and an intervening variable (e.g., agricultural production) against an aggregate indicator of SC that comprises the five indicators noted above.

Political scientists who advocate interpretivist or postmodern analyses may reject the utility of attempts to quantify SC, particularly using this set of economic, demographic, and social indicators. However, the sociologist Jack Goldstone argues that empirical social indicators are important in revealing the nature of societal instability, and that economic and demographic pressures have been the core sources of rebellion and revolution over the centuries.[10]

In this book, I adopt the perspectives that Goldstone and Homer-Dixon adopted in their attempts to measure the associations between empirical indicators and SC and, beyond that, to employ qualitative means to determine the causal linkages between variables. However, some aspects of Homer-Dixon's model of SC remain difficult to quantify, particularly instrumental rationality and coherence. It may be possible to explore such

non-quantifiable aspects of SC during the case studies that will eventually follow. Perhaps social scientists will need to develop new types of quantitative indicators in order to explore the mathematical associations between these aspects of SC and the various parameters that drive these dependent variables.

**The Independent Variable**

For the purposes of this book, I provide a specific definition of ERIDs: pathogen-induced human illnesses that have increased in incidence, lethality, transmissibility, and/or expanded their geographical range since 1973.[11] Specifically, this includes previously unknown pathogenic agents such as HIV, *E. coli* 0157 H7, Ebola virus, hantavirus, prions, hepatitis A–C), and antibiotic-resistant pathogens such as vancomycin-resistant enterococci and methycillin-resistant *Staphylococcus aureus*. Re-emerging diseases are those pathogen-induced human illnesses that were previously controlled or declining in range and/or incidence but are now expanding in range, incidence, drug resistance, and increasing transmissibility and/or lethality. Some of the re-emerging diseases are tuberculosis, malaria, cholera, dengue fever, yellow fever, schistosomiasis, rotavirus, adnovirus, and amebic dysentery. Pathogens are defined as viral, bacterial, parasitic, or proteinic organisms or agents that live in a parasitic and debilitating relationship with their human host.

It is important to think of pathogens as exogenous variables—natural agents that for the most part exist independent of humanity and for all intents and purposes have one central goal, survival. As zoonoses, microbes have historically crossed over from disease pools that exist in animal reservoirs.[12] Human activity frequently alters flows of viral traffic, and these novel pathogens may subsequently take root within the human ecology. The major human pathogens listed in table 1.2 qualify as ERIDs under this definition.

These various microbes and parasites constitute the majority of pathogens that generate significant morbidity and/or mortality in human beings. Intending to test the effect of these diseases on state capacity, I would want to employ detailed state-specific information on national prevalence rates. In an optimal scenario, standardized prevalence rates for

each major disease would be available for each and every country, from 1950 to the present day. Owing to the many measurement problems detailed below, such data are not available. Since very few states collect comprehensive and composite national pathogen-specific disease prevalence indicators, we must employ proxies that measure the overall burden of disease on selected states in the sample.[13]

For many of the developing countries, even basic mortality and morbidity data are lacking. For example, many countries do not collect malaria prevalence on an annual basis; in fact, the World Health Organization has stopped collecting plasmodium prevalence statistics for all of sub-Saharan Africa because the disease is ubiquitous. Furthermore, some countries (e.g., China and Myanmar) fail to report the occurrence of certain diseases (such as HIV) owing to a lack of political transparency. (Certain governments go to great lengths to keep information about human-rights abuses and other domestic matters from being documented and to keep such information from being released to the world at large.) To complicate matters further, many diseases carry a social stigma, and physicians are often pressured into falsifying diagnoses in order to "preserve face" for the afflicted. The greatest difficulty arises from the fact that in some countries reports of disease incidence are often sporadic, and often only cover a few of the many diseases that compromise the health of that state's population. All told, these problems make it extremely difficult for the analyst to conduct a statistical analysis of the aggregate effect of infectious diseases on the stability and productivity of a society (i.e., its state capacity).[14] However, limited incidence and prevalence rates are available for selected diseases in certain industrialized countries. These data sources are listed in table 1.3, which also indicates their availability.

Given these measurement problems, the social scientist must employ indicators that serve as comprehensive proxies to measure the burden of disease-induced morbidity and mortality on societies. Such an indicator must be highly sensitive to the societal burden of disease, it must be standardized across states, it must be available for most countries over a broad span of time, and it must be comprehensive (such that it reflects mortality associated with the most prevalent pathogens in a society). In view of these requirements, the most valuable comprehensive empirical indicators

**Table 1.2**
Pathogenic agents and associated diseases.

| |
|---|
| Human immunodeficiency virus (AIDS) |
| *Mycobacterium tuberculosis* (tuberculosis) |
| *Plasmodium malariae, falciparum, vivax, ovale* (malaria) |
| Hepatitis A, B, and C viruses |
| *Vibrio cholerae* (various subtypes, notably El Tor) |
| Flavivirus DEN-1,2,3,4 (dengue fever) |
| Filoviruses (e.g., Ebola virus) |
| *Escherichia coli* 0157 H7 |
| *Flaviviridae* viruses (yellow fever) |
| *Phlebovirus bunyaviridae* (Rift Valley fever) |
| *Schistosoma mansoni, haematobium, japonicum* (schistosomiasis) |
| *Onchocerca volvulus* (river blindness) |
| *Mycobacterium leprae* (leprosy) |
| *Dracunculus medinensis* (dracunculiasis) |
| Hantaviruses (hantavirus pulmonary syndrome) |
| *Leishmania chagasi* (leishmaniasis) |
| *Shigella dysentaeraie, flexneri, boydii, sonnei* (shigella) |
| *Corynebacterium diptheriae* (diphtheria) |
| Rotavirus (severe diarrhea) |
| Respiratory syncytial virus and parainfluenza virus type 3 (ARV) |
| Adnovirus (severe diarrhea) |
| *Legionella pneumophila* (Legionnaires' disease) |
| *Cryptosporidium parvum* (cryptosporidosis) |
| Human T-lymphotropic virus 1 and 2 |
| Toxin-producing strains of *Staphylococcus aureus* ("flesh-eating disease") |
| *Borrelia burgdorferi* (Lyme disease) |
| Prion proteins (Creutzfeldt-Jacob disease) |
| *Helicobacter pylori* |
| *Enterocytozoon bieneusi* |
| *Cyclospora cayetanensis* (cyclosporosis) |
| Herpesvirus simplex 1 and 2 (herpes) |
| *Treponema pallidum* (syphilis) |
| *Haemophilus influenzae* type B (meningitis) |
| *Ehrlichia chaffeensis* (ehrlichiosis) |
| *Encephalitozoon hellem* |
| *Bacillus anthracis* (anthrax) |
| *Tyrpanosoma cruzi* (Chagas' disease) |
| *Bartonella hemsellae* |
| *Encephalitozoon cuniculi* |

**Table 1.2 (continued)**

| |
|---|
| *Neisseria gonorrhoae* (gonorrhea) |
| Influenza virus A, B, C (flu) |
| *Bordetella pertussis* (pertussis) |
| *Borrelia burgdorferi* (Lyme disease) |
| *Salmonella typhi* (typhoid) |
| *Yersinia pestis* (plague) |
| *Chlamydia trachomatis* (chlamydia) |
| *Trypanosoma brucei rhodesiense, gambiense* (African sleeping sickness) |
| Arenavirae; sub-types Tacaribe, Junin, Machupo, Lassa, Guanarito, Sabia (viral hemorrhagic fevers) |
| *Clostridium botulinum* (botulism) |
| *Camplyobacter jejuni* |
| *Giardia lamblia* |
| *Entamoeba histolytica* (amebic dysentery) |
| Filarial nematodes (filariasis) |

are *infant mortality per 1000 children* and *life expectancy (in years) at birth*. Data for these two proxies are available for the vast majority of countries over the period 1950–1991.

Infant mortality (IM) is arguably the best indicator for measuring the aggregate burden of disease on a population, as it incorporates mortality from every disease pathogen, including syncytial respiratory viruses, diarrheal rotaviruses and adnoviruses, and the pathogens that cause malaria, tuberculosis, and measles. IM measures the effect of infectious disease on the first tail of the demographic distribution of a population: children up to age 5. With the notable exception of HIV/AIDS, IM is the best indicator for measuring the burden of disease across divergent societies because the majority of global disease-induced mortality shows up in the 0–5-year age sector of the demographic curve. In layman's terms this means that the vast majority of human beings killed by infectious diseases are children below the age of 6. Indeed, Murray and Lopez demonstrate that more than 70 percent of global infant mortality is attributable to microbial and/or parasitic infection.[15] Therefore, fluctuations in IM over time typically result from the changing prevalence and lethality of infectious diseases within specified populations.

**Table 1.3**
Availability of disease data.

| | | |
|---|---|---|
| US | HIV/AIDS, anthrax, botulism, brucellosis, chlamydia, cholera, diptheria, *E. coli* 0157 H7, gonorrhea, leprosy, encephalitis, hepatitis (A,B,C), legionella, Lyme disease, malaria, measles, pertussis, plague, salmonellosis, shigellosis, streptoccal disease (invasive group A). | Weekly state and territorial disease-specific incidence data available since 1984[a] |
| Australia | HIV/AIDS, malaria, hepatitis (B,C), tuberculosis, cholera, diptheria, dengue fever, typhoid, syphilis | Annual national prevalence data available since 1985[b] |
| France | Diarrheal diseases | 1990–1999 |
| | Influenza | 1984–1999 |
| | Measles | 1985–1999 |
| | HIV | 1989–1999[c] |
| Canada | HIV, salmonella, influenza, tuberculosis, hepatitis (B,C) | Incidence by province/month 1995–1999[d] |
| Global aggregate | HIV seroprevalence | 1997,[e] 1998, 1999, 2000 |

a. Statistics for these diseases are available for the US in Morbidity and Mortality Weekly Report. Stats are given in weekly, and four week totals, according to state and overall national incidence. National prevalence levels are not similarly available for these years. See http://www.cdcc.gov/epo/mmwr/mmwr.html and http://www.cste.org.
b. See http://www.health.gov.au/pubhlth/cdi/nndss/year013.htm and http://www.avert.org/canstatr.htm.
c. Available in graph format at http://www.b3c.jussieu.fr/sentiweb. Exact values are not given.
d. See Health Canada's Communicable Disease Reports 1995–2000 at http://hwcweb.hwc.ca/hpb/lcdc/publicat/ccdr.
e. Current estimates of overall HIV prevalence at the national level for 1997 are regarded as reasonably accurate. See http://www.unaids.org/highband/document/epidemio/june1998/fact_sheets/pdfs/botswana.pdf.

Nowhere in the literature can one find a specific statement that IM is a good proxy for a comprehensive snapshot of the burden of infectious diseases on societies. This is so because medical scientists usually study a single disease pathogen and because they have never thought about the relationship between disease and state capacity. In other words, they have never had to argue that IM is a good comprehensive indicator, although most medical scientists I have spoken with agree that this is an empirically valid claim.[16]

Murray and Lopez quantify the aggregate burden of disease by including both long-term and sporadic morbidity, and disease-induced mortality. They use the measurement tool of the disability-adjusted life year (DALY) in order to look at the true effects of various diseases, injuries, and risk factors on affected populations. "DALYs," they note, "provide a common metric to aid meaningful comparison of the burden of risk factors, diseases, and injuries," and "the primary indicator used to summarize the burden of premature mortality and disability (including temporary disability) is the disability-adjusted life year (DALY)." "DALYs," they explain, "are the sum of life years lost due to premature mortality and years lived with disability adjusted for severity."[17]

Murray and Lopez demonstrate that the top two contributors to the global burden of disease are communicable diseases affecting children, namely lower respiratory infections (LRIs) and diarrheal diseases primarily caused by the adno and rotaviruses and amebic agents. Tuberculosis, measles, malaria, and pertussis also came in as the seventh, eighth, eleventh, and twenty-third greatest contributors to global death and disability, and all these illnesses are found at relatively higher levels in the youngest tail of the population curve, namely the 0–5-year age group. Murray and Lopez note that infectious disease constitutes the single greatest burden on human populations relative to all other causes of death: ". . . the three leading contributors to the burden of disease are lower respiratory infections, diarrheal diseases, and perinatal disorders. Together with measles, the eighth largest cause of burden, these childhood diseases account for 25 percent of the whole burden of premature mortality and disability."[18]

Disease-specific DALY measurements for all states over a significant period of time would be tremendously valuable to this type of project.

However, until this information is available we must use proxies that are extremely sensitive to the burden of disease. Thus, IM's sensitivity to the comprehensive societal burden of disease makes it the best available indicator for measuring the effects of ERIDs on state capacity over broad stretches of time and across a wide range of cultures and societies.[19]

However, IM will not include the entire burden of certain pathogens (such as HIV) that predominantly affect the central part of the demographic distribution curve, namely those in the 15–45-year age range. *Life expectancy* (LX) measures the total burden of disease on a specified population, covering the complete demographic curve (including both tails of the population distribution). Unfortunately, the mortality shown under LX does not replicate IM's extreme sensitivity to infectious disease, as it includes mortality resulting from accidents, suicides, and violence. However, rapid increases in the prevalence of HIV within a society will show up only in the 15–45-year portion of the demographic distribution. Thus, although the effects of the HIV pandemic on national productivity and stability are unlikely to show up in IM, they may be observed through the use of LX. "From independence in 1980 and for nearly a decade thereafter," Madavo writes, "Zimbabwe made stunning health advances. But AIDS has already erased all the life expectancy gains made since then. Further, if the worst projections come to pass, by about 2010 life expectancy will return virtually to where it stood the day I was born, in what was then Southern Rhodesia, half a century ago."[20]

Preston's detailed international statistical analyses of the major causes of mortality decline are valuable in determining the major causes of death over time. Preston expanded on previous work done with Keyfitz and Schoen[21] to examine the relative importance of various causes of death using data for 165 populations from various countries and across various time periods.[22] Preston found that in the twentieth century at least 60 percent of global mortality was in fact attributable to infectious disease. Vallin notes that Preston's estimates of the influence of disease on mortality were on the low side, as the remaining 40 percent of mortality was attributed to ill-defined causes and did not include pathogen-induced cancers. Vallin et al. also attribute considerable weight in diachronic measures of global mortality to infectious diseases, and provide evidence that reinforces Preston's conclusions.[23] Of course, variation will occur in the

causes of death between different populations and over time, but diachronic and randomized statistical studies such as this should minimize those possible skewing effects. LX is therefore required as a supplementary indicator to give us an accurate picture of the effects of the HIV pandemic.

Recent medical advances have shown that some pathogens play major roles in inducing many forms of human cancer. "Up to 84 percent of cases of some cancers are attributable to viruses, parasites or bacteria. WHO estimates that more than 1.5 million (15 percent of the new cases occurring each year) could be avoided by preventing the infectious disease associated with them. About 1.2 million cancer cases (20 percent) in developing countries and 363,000 (9 percent) in developed countries are attributable to infectious agents."[24] These cancers include stomach cancers (*Helicobactor pylori*), cervical cancers (human papilloma virus), liver cancers (hepatitis B and C), AIDS-related cancers (numerous pathogens), Burkitt's lymphoma (Epstein-Barr virus), Hodgkin's disease (Epstein-Barr virus), and bladder cancer (schistosomiasis).[25] Therefore, LX compliments IM because it measures mortality resulting from both the global HIV pandemic and pathogen-induced cancers.

LX displays an inverse statistical association with IM, such that there is a significant negative correlation (–0.935) between IM and LX for the 20-country sample over the period of the analysis (1950–1991). Thus, it can be stated with assurance that IM and LX are generally "mirror proxies" that both measure the burden of disease on populations but have an inverse relation to one another. This is useful because these indicators allow us to analyze the burden of disease on the complete demographic distribution of the population within a state. IM and LX also provide us with a comprehensive snapshot of the burden of disease over a relatively broad span of time and reflect the decline over time in morbidity and mortality as measured since the early 1950s, which can then be measured against changes in state capacity over the same time period.

One caveat in regard to the use of IM and LX as proxies for ERIDs is that, although they give us an excellent idea of ERID-induced *mortality* over the decades, they only give us indirect knowledge of the *morbidity* associated with ERIDs over the same time period. For example, malaria-induced mortality will show up in IM, but we can only guess at the ratio of individuals killed as to the proportion of the population that

is debilitated. This also varies according to the lethality of the disease, as malaria generally debilitates far more people than it kills, whereas HIV generally debilitates and kills those who it affects. Regrettably, a majority of states lack the ability to accurately track disease-induced morbidity within their populations. Therefore, I cannot employ the 20-country sample to ascertain the effect of specific disease-induced morbidity on state capacity. At this point in time, only a very small subset of industrialized nations (Canada, the United States, the United Kingdom, Australia) keep limited statistics on productivity lost to diseases such as HIV. Obviously, studies that measure morbidity and mortality-generated DALYs (e.g., Murray and Lopez's *Global Burden of Disease*) will be of enormous value if differentiation of pathogen weight by country per year is included in future editions. Notably, the WHO has begun to publish reasonably accurate statistics on HIV seroprevalence rates within national populations for most countries of the world for the year 1997.[26]

## Theories of Causation

> There is . . . a good deal of evidence that bacteria became capable of producing infections millions of years ago, and there is no reason to doubt that man from the very beginning suffered from infectious disease; and at the time when mankind had reached the period of the earliest historical records, infectious diseases of many varieties already existed. . . .
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> —Hans Zinsser, *Rats, Lice, and History,* p. 106

Microbial pathogens evolved from the primordial soup of life millions of years ago, along with other single-cell creatures, and thus have existed far longer than humans (much less human societies), preying on all manner of flora and fauna over the eons.[27] Thus, pathogens predate humanity, tend to exist independent of humanity in nature, and will continue to exist whether the human species endures or not. Therefore, pathogens should be seen as independent phenomena that can be affected by human actions that may then alter microbial transmissibility and lethality.

There has been some debate regarding the lines of causation in the complex relationship between ERIDs and state capacity. The principal objection voiced is that infectious disease is in fact *endogenous* and therefore

caused by pre-existing human-induced conditions such as poverty, war, famine, and environmental degradation.[28] The fact of the matter is that these social conditions are actually *intervening variables* that (depending on their individual nature) may increase the transmission capacity, the infectivity, or the lethality of pathogenic agents within affected regions. However, the argument that these conditions *create* the pathogens in question is incorrect. There is significant archeo-epidemiological evidence that infectious pathogens antedated the arrival of humans (and multi-cellular life in general) and that their rapid and unpredictable evolution is guided to a large degree by complex and chaotic ecological interactions and is occasionally accelerated by human actions.[29]

The concept of pathogen emergence is critical, since new disease agents tend to exhibit the greatest virulence when first introduced to immunologically naive populations. To paraphrase Morse and Schluederberg: "Emerging" pathogens are disease agents that either have recently appeared in the population or are rapidly expanding their range.[30] Morse argues that known disease agents are "only a fraction of the total number that exist in nature."[31] Furthermore, "newly evolved" disease agents are most often the descendants of antecedent strains; this is a function of Darwinian evolution through processes of natural selection. "Given these constraints of organic evolution, then, there are fundamentally three sources (which are not necessarily mutually exclusive): (1) evolution of a virus *de novo* (usually the evolution of a new viral variant); (2) introduction of an existing virus from another species; (3) dissemination of a virus from a smaller population in which the virus might have arisen or originally been introduced."[32] Similar processes also hold for bacteria, for parasites, and perhaps for infectious proteins (prions).

However, according to Morse, pathogen evolution is not the most significant driver behind the emergence of "new" infectious diseases: ". . . over the period of recorded history . . . 'emerging viruses' have usually not been newly evolved viruses. Rather, they are existing viruses conquering new territory. The overwhelming majority are viruses already existing in nature that simply gain access to new host populations."[33] These pathogens exist in nature in disease "reservoirs" and may jump the species barrier to humanity from the "zoonotic pool" (i.e., the vast

plethora of diseases that pervade all niches of life in the biosphere). Although the chance that any one particular "zoonosis" is pathogenic to humans is relatively low, the sheer magnitude of infectious agents that exist in the zoonotic pool makes the "emergence" of human pathogens more likely. Morse coined the term *viral traffic* to demonstrate how infectious agents move between species and between individuals, and he argues that most outbreaks of "new" diseases are attributable to patterns of viral traffic. Viral traffic is altered by changes in the ecological, economic, and social environment. I refer to such changes as *facilitating variables,* insofar as they may exacerbate the lethality and the transmission of ERIDs and thereby intensify the negative effects of ERIDs on state capacity.

**Intervening Variables**

> The spread of [leishmaniasis] is accelerated by development programs such as road building, dam construction, mining and forest exploitation that bring increasing numbers of people into contact with the disease vectors. Another factor enhancing spread is the haphazard growth of major urban centers which creates conditions that increase transmission risks. A third factor is the movement between countries or regions of migrant workers who themselves act as vehicles for the disease.
>
> —*World Health Report 1996,* p. 50

It is important to keep in mind that the effects of infectious disease on state capacity are distinctly nonlinear, as pathogens are subject to such intervening variables as ecological disruption (chapter 4), increased human mobility, poverty, technology, war, and famine. These factors often alter the flow of viral traffic, thereby producing epidemics and pandemics and affecting their courses. In this way, these intervening variables act as *disease amplifiers*. Augmentation of the virulence and the transmissibility of pathogens by these disease amplifiers generates epidemic and/or pandemic disease. These facilitating variables generally exacerbate the ERID threat, but it is important to understand the dynamics between ERIDs and these facilitating variables, as they frequently influence one another in a complex web of mutual and nonlinear interactions.[34] These interactions require the fulfillment of certain conditions that, together, are sufficient to produce ERIDs. Here I will list these facilitating variables briefly, in descending order of importance.

**Migration**

International and intra-state migration is playing a significant role in the diffusion of pathogens. Travelers to and from previously isolated regions may distribute previously contained microorganisms into the global population, many of whom will be immunologically naive to the emerging infectious agent. Furthermore, travelers from the developed countries bring pathogens from their sojourns abroad back into their home countries where these agents may eventually take hold within that new population. Rapid advances in transportation technologies (the ship, railway, car, airplane) have accelerated this process of global pathogen diffusion, and the profusion of international travelers for both recreational and business purposes is bound to exacerbate the problem of ERID dissemination in the coming decades.[35]

**Trade**

Throughout history, trade has been implicated in the worldwide diffusion of pathogens. For example, both flavidirae viruses (e.g. yellow fever) and their principal vectors (*Aedes aegypti* mosquitoes) were transmitted to the Americas from Africa courtesy of the slave trade. The mosquito vectors fed on the blood of infected slaves during the transit and then spread the contagion throughout the New World.[36] Additionally, the Pan American Health Organization believes that the recent transmission of El Tor cholera to South America was facilitated by a Chinese freighter which jettisoned its contaminated bilge water into a Peruvian harbor, after which the disease spread through seafood products and tainted regional water supplies.[37] Additionally, infected foodstuffs and livestock transported across borders have resulted in dissemination of the BSE prion to beef cattle throughout the European Community. Infected berries (cyclospora) from Guatemala were implicated in a large outbreak of diarrheal disease throughout North America during the summer of 1996.

**Human Ecology**

The actions of individuals within a society, and societal habits at large can also influence the course of viral traffic and lead to the emergence and reemergence of infectious disease, both regionally and globally. For example, the annual pilgrimage to Mecca is generally associated with the

proliferation of cholera among the pilgrims, who then bring the bacilli back to their home countries. Other modes of behavior, particularly sexual promiscuity and the use of illicit narcotics, assist in the diffusion of many disease agents. Furthermore, the burgeoning magnitude, density, and distribution of human populations facilitates the dissemination of pathogens—particularly since, once population levels reach a new threshold, "disease pools" within those populations become large enough to sustain new infections.[38]

**Misuse of Antimicrobial Drugs**

Consistent misuse of antimicrobial drugs in developed and developing countries has resulted in the emergence of drug-resistant strains of parasites, bacteria, and viruses. For example, the Thai-Burmese border region is practically uninhabitable owing to the recent spread of drug-resistant strains of malaria throughout the region. Meanwhile, in the developed countries, bacterial strains such as vancomycin-resistant enterococci and methycillin-resistant *Staphylococcus aureus* are spreading throughout hospital systems, and multi-drug-resistant tuberculosis is spreading through the marginalized portion of the population. The problem stems from the fact that organisms develop drug resistance through evolutionary pressures when the pathogens in question are exposed to antimicrobial drugs. These antimicrobial agents kill susceptible bacteria, which in turn generates evolutionary pressures on those members of the species that possess a gene that provides resistance to that particular drug. These resistant microbes then expand their population to fill the ecological niches of other pathogens that were eradicated by the same antimicrobial agents.[39] Although physicians use the current drug of last resort (vancomycin) extremely sparingly, tremendous amounts of similar drugs are distributed through domestic animal feed, which results in the spread of resistant bacteria throughout the animal world. These resistant pathogens may then cross the species barrier to cause zoonoses in human populations.

**Disasters**

In addition to the above facilitating variables, both natural and human-induced disasters (e.g. earthquake, flood, war, famine) may also affect viral traffic in a manner that leads to disease amplification through increased transmission and/or lethality of the infectious agents and which may result

in epidemics and even pandemics. The worst case of this occurred during 1918 and 1919, when the global movements of armies served as vectors for the distribution of influenza and typhus. The resulting "Spanish flu" pandemic claimed an estimated 20 million lives, and the typhus pandemic claimed nearly as many, dwarfing the mortality caused by military action.[40]

Similarly, a regional breakdown of food distribution that results in famine will also deplete the health of a population, such that infectious agents may have an easier time colonizing their hosts and may cause greater morbidity and mortality in the weakened population, as it takes the weakened host a longer time to mount an effective immune response to the invading pathogens. The greatest historical example of this synergy between famine and disease is the Great Hunger that struck Europe in 1845. This catastrophe was generated by a fungus (*Phytophthora infestans*) that destroyed the potato crops, caused massive starvation and governance problems in Ireland, and led to terrible outbreaks of typhus and cholera in affected regions, which were subsequently carried overseas to North America and Australia via infected immigrants who fled the devastation in the Old World.[41]

**Data**

The data I use in this analysis are taken from a random sample of 20 countries: Botswana, Brazil, Colombia, Ethiopia, Haiti, Iceland, India, Italy, Japan, Kenya, Malawi, Netherlands, Norway, Peru, Rwanda, Saudi Arabia, South Africa, Tanzania, Thailand, and Uganda. The country data used for the statistical analysis are from the World Bank Statistical Tables data set, the WHO's World Health Reports, and the UNAIDS statistical country fact sheets. Primary and secondary source epidemiological and microbiological data were obtained from the Population and International Health, and Countway Libraries at the Harvard School of Public Health. I have also used the ProMed global disease surveillance system, the US Bureau of the Census HIV Surveillance sentinel site data base, the World Health Reports, the Morbidity and Mortality Weekly Reports, journals such as *Science, Nature, The Economist,* the *Journal of the American Medical Association, New England Journal of Medicine, The Lancet, Emerging Infectious Diseases,* and numerous health-related Internet sites[42] as core data sources.

For the purposes of the quantitative analysis, I employ standardized diachronic global indicators of state capacity, such as GNP per capita and school enrollment. These measures run from circa 1960 to 1991 and are all obtained from standardized World Bank data. IM and LX data are generally available for the full sample from 1952 to 1991.

The data I use allow for the variation of both dependent and independent variables while allowing for some control over potentially confounding variables. In order to avoid selection bias in the analysis, I have randomly selected cases hoping for significant variance in the independent variable between the countries in the sample. Thus, I compare indicators from highly developed temperate states such as Iceland (with low ERID intensity) with those of tropical countries like Rwanda (with high ERID intensity). This eliminates the bias that might result from selecting only developing countries with low state capacity.

Randomizing the selection process is crucial to reducing bias. Before randomization, certain states had to be selected out of the population for inclusion in the sample because they could not satisfy the minimum data requirements. Countries such as Sierra Leone and Liberia that lacked a minimum standard of data for the selected indicators were excluded from the sample because their inclusion would have been of low utility. The sample was drawn randomly from the remainder of countries that met the minimum data requirements for the relevant indicators, even if there were occasionally significant gaps in the annual data. This randomization was generally successful, as the countries in the sample represent all climatic regions of the world, all levels of development, and most continents. This random selection of the sample should suffice to reduce the probability of bias to a reasonable minimum. Furthermore, the size of the sample ($N = 20$) is also sufficient to do a good job in terms of obtaining a "snapshot" of the correlations between the variables on a comparative cross-national basis.

Despite the advances of modern ERID surveillance technologies, there remain data problems that other scientists must consider and try to circumvent. First, a lack of transparency frequently hinders the collection of accurate field data and the dissemination of accurate statistics within the country in question. As well, political barriers may rise in order to hide the true state of affairs (i.e., the massive debilitation of the population by a stigmatized disease such as HIV/AIDS). This was commonplace with HIV/

AIDS prevalence in sub-Saharan Africa in the 1980s, and throughout South and East Asia in the 1990s. The intra-state dissemination of accurate statistics is also problematic because of the lack of technological diagnostic and communication infrastructure, and manpower, in many of the rural hospitals. Furthermore, regional authorities may have interest in exaggerating the infectious disease situation in order to receive greater amounts of aid, or conversely downplaying the gravity of the situation to avoid unfavorable reviews from their superiors and to prevent the loss of revenue from trade and tourism. This political manipulation and suppression of accurate statistics makes it difficult to get accurate disease-specific data out of many states, particularly those with authoritarian regimes (such as Zimbabwe, China, Myanmar, and the former Zaire). The greater political transparency of democratic nations allows better data collection.

At the systems level, data collection is improving as nascent global surveillance regimes such as ProMED report outbreaks and occurrences daily via electronic media. As mentioned above, the World Health Organization and the US Centers for Disease Control issue quarterly reports on the prevalence of certain notifiable infectious pathogens, and weekly updates such as the Morbidity and Mortality Weekly Report offer tallies of disease incidence within the US population. However, for the majority of the population of states the data are marred by certain inaccuracies at this level as well. First, it is very hard to obtain accurate data on the incidence of and/or prevalence levels for certain pathogens (e.g., those that cause hepatitis) within specified national populations (e.g., Sierra Leone). Sentinel data are available for selected diseases in selected communities on various dates, but it is difficult at best to derive national seroprevalence levels from these scattered studies.[43] Second, in some cases the agencies that are expected to monitor prevalence levels have simply bowed to the enormous prevalence in certain regions and stopped collecting data on selected pathogens. This is the case in sub-Saharan Africa, where the WHO has admitted that it no longer has the capacity to monitor the prevalence of malaria. Finally, there are occasional scientific inconsistencies in the collection, interpretation, and dissemination of data from the various reporting sites to the WHO. Though the WHO attempts to harmonize the data as much as possible, it is likely that some inaccuracies will remain in the data. As local, regional, state, and WHO infrastructure improves over time, the data will improve.

Despite certain deficiencies noted above, it is possible to derive generalizable and empirically testable scientific hypotheses from the data. To ensure that my conclusions are simultaneously demonstrable and accurate, I employ statistical data analyses. Statistical analysis of empirical data provides correlations that confirm or disconfirm the various hypotheses, and it allows me to discriminate between potentially important causal linkages and ones that are marginal to the subject at hand. Quantitative techniques can indicate whether there are any potentially causal relationships.

I employ bivariate statistical analysis, using Pearson's correlation coefficient to test the strength of the hypothesized relationships between the variables. The correlations that I derive from these tests indicate the strength of the association between two variables between the values of 1 and –1, and they give the significance of the association in view of the size of the sample. Significance indicates a real and important relationship between the variables and suggests that these findings can be generalized to the entire population.

The use of *t* tests (tests of statistical significance) on Pearson's *r* (the correlation) tells whether the correlation differs significantly from 0. The above tests can either support or refute the respective hypothesis with 95 percent confidence. The margin of error based on sample size is ±10 percent. Regardless of the sample size, an $\alpha$ of at least 0.6 is required before it can be said that there is a strong correlation among the observed variables that constitute the computed SC variable. Having computed $\alpha$ as 0.64, we can attest to the firm inter-correlation between the five SC indicators that constitute the aggregate SC measure.

These statistical analytic processes can tell us much, but they cannot firmly specify the nature of the causal relations within the model. Initially, I correlate the independent and dependent variables for the entire period 1950–1991. I then lag the variables to see if the strength of the correlation changes downstream. Theoretically, disease-induced mortality and morbidity are likely to impair state capacity, but this effect is not likely to be immediate. For example, after colonizing a human host, pathogens often take differential amounts of time to generate disease within that host. Thus, the debilitating effects of certain diseases with long germinating periods (such as HIV/AIDS, hepatitis B, and hepatitis C) will logically grow stronger with the passage of time. Therefore, I lag the variables to see if the

downstream effect of disease on state capacity grows or diminishes with the passage of time.

Infectious diseases' pronounced negative effect on child life expectancy will reduce the downstream availability of healthy and capable workers available to a society. By lagging the variables, I analyze the import of the differential time lag between increasing ERID values and state capacity outcomes. This will help in the prediction of downstream economic and political effects of rising disease levels, in the formulation of more effective policy measures to deal with the problem of infectious disease, and in the prediction of downstream state capacity on the basis of current population health indicators.

Since the lack of sufficient quantifiable data rules out factor analysis of the relationships among the variables, it is necessary to explore the question of causality using available non-quantifiable data. Process-tracing case studies make it possible to distinguish spurious correlations from probably causal relationships, and can help us get a handle on certain interactions that are difficult to correlate because of operationalization and measurement problems. Mapping the complex threads of causation among the independent, intervening, and dependent variables illuminates the probable causal connections among them. For example, although statistical data analysis may demonstrate a high correlation between the burden of disease on a society and that society's productivity, scientists must utilize process-tracing techniques to determine the causal linkages between the variables and the appropriate mechanisms of causation.

**Falsifiability**

The hypothesis that increasing infectious disease prevalence diminishes state capacity is easily falsifiable. It will be shown false if increasing levels of disease do not correspond to declining state capacity or if falling infectious disease rates do not correlate with increasing state capacity. Since most of the available data are from the most successful anti-microbial era in human history (1950–1991), one can test the hypothesis empirically by looking at how declining disease-induced morbidity and mortality has affected state capacity since World War II. If the inverse relationship holds between proxies for disease prevalence and state capacity, one can

generalize that the negative association between infectious disease and state capacity will also hold over time and across geographical regions. Thus, if the hypothesis is correct, one can argue that the expanding pandemics of HIV/AIDS, tuberculosis, malaria, and hepatitis (to name just a few) will have negative implications for state stability and development in the future.

In view of the theorized empirical centrality of disease as a stressor on state capacity, any major threat to a population's health jeopardizes a state's prosperity, governance, and survival over the long term. Thus, infectious disease can be seen as a significant factor in the breakdown of governance, poverty, and state failure in seriously affected regions. Conversely, and of equal importance, declining disease rates should lead to greater state capacity and, by extension, to greater prosperity, stability, and power in healthier areas, such as the temperate zones.

My analysis of the effect of infectious disease on the populations of states and its resultant effect on states' prosperity and stability obviously has broad ramifications for most (if not all) human societies. If we can understand the relationship between rising levels of ERIDs and the associated decline in prosperity and stability of states and societies, then we gain the ability to address the break points in the chain of causation in order to formulate more effective policies for the surveillance and containment of infectious disease. We may also gain some ability to predict future events and processes that may be detrimental to a state, such as disease-related socio-economic decline, insurrection, rebellion, and (in extreme cases) state failure.

Population health is a significant *parameter* of state capacity. A parameter is defined as a phenomenon that exerts a general effect on another dependent phenomenon. In this sense, broadly defined constructs such as population, environment, poverty, and (in particular) population health may generate significant positive or negative effects on state capacity. The central concerns of any study of parameters of state capacity are to determine the importance of each parameter relative to the others and to determine how each parameter affects state capacity. Bias-free systematic studies of the other parameters will have to be completed before it will be possible to determine the relative weights of the various parameters as they affect state capacity.